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CEREBRAL HYPOXIA DURING OVERLOADS IN THE CEPHALO-PELVIC DIRECTI--ETC(U)
JAN 79 Y A KOVALENKO, V L POPKOV

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by

Ye.A. Kovalenko, V.L. Popkov, and
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EDITED TRANSLATION

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Г г	*Г г*	G, g	У у	*У у*	U, u
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К к	*К к*	K, k	Ъ ъ	*Ъ ъ*	"
Л л	*Л л*	L, l	Ы ы	*Ы ы*	Y, y
М м	*М м*	M, m	Ь ь	*Ь ь*	'
Н н	*Н н*	N, n	Э э	*Э э*	E, e
О о	*О о*	O, o	Ю ю	*Ю ю*	Yu, yu
П п	*П п*	P, p	Я я	*Я я*	Ya, ya

*ye initially, after vowels, and after ъ, ь; e elsewhere.
When written as ё in Russian, transliterate as yё or ё.

RUSSIAN AND ENGLISH TRIGONOMETRIC FUNCTIONS

Russian	English	Russian	English	Russian	English
sin	sin	sh	sinh	arc sh	$\sinh^{-1}$
cos	cos	ch	cosh	arc ch	$\cosh^{-1}$
tg	tan	th	tanh	arc th	$\tanh^{-1}$
ctg	cot	cth	coth	arc cth	$\coth^{-1}$
sec	sec	sch	sech	arc sch	sech^{-1}
cosec	csc	csch	csch	arc csch	csch^{-1}

Russian	English
rot	curl
lg	log

CEREBRAL HYPOXIA DURING OVERLOADS IN THE CEPHALO-PELVIC DIRECTION

Ye. A. Kovalenko, V. L. Popkov, and
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Moscow

The study of the effect of overloads on an organism was begun at the end of the last and the beginning of this centuries (Salathe 1877, Tsibul'skiy 1879, Pashutin 1881, Nemzer 1892, Voyachek 1908, and Tsiolkovskiy 1912). N. O. Tsibul'skiy and V. V. Pashutin have expressed an idea with regard to a reflex regulation of blood circulation when an organism is affected by gravitational forces caused by the change in the body position of an animal. This idea was developed further in the works of I. R. Petrov (1949, 1952), who showed the presence of compensatory reactions at small overloads even in animals which were weakened by blood loss. These protective reactions permit the animal to maintain the arterial pressure at a relatively high level for a certain period of time (stress of 1 g in the cephalo-pelvic direction) while the animal is immobilized in a vertical position.

An extensive experimental study of the effect of overloads on an organism began together with the arrival of high-speed aircraft. Flights on such aircraft required the development of protective "antioverload" means. This problem could only be solved only under the condition of a thorough study of the mechanism by which the overloads act on the man's organism.

It was shown in numerous studies that sharp changes occur in

hemodynamics and breathing under the effect of overloads (Jongbloed, Noyons, 1933; Henry et al., 1951; Babushkin and co-authors, 1958; Langdon, Reynolds, 1961).

In particular, during the head-pelvis overloads, there is a drop in blood pressure in the vessels of the orbital floor and carotid artery, reaching a zero level at significant overloads (Akesson, 1948; Duane, 1954; Hershgold, steiner, 1960). This occurs as a result of the fact that the hydrostatic blood pressure in the head-heart region increases by 10 times at a 10-fold overload. Naturally, the heart cannot push the blood to the head under such pressure and, in this case, one should expect a complete cessation of blood supply to the brain. However, in observing the blood flow in the vessels of the brain through a special opening in the cranium, blood flow was observed in these vessels by means of color photography, even when the blood pressure in the carotid artery was reduced to zero (Jasper, Cipriani, 1945). Under these conditions, the preservation of blood supply to the brain is explained by the fact that, in parallel with a decrease of blood pressure in the carotid artery, there is a progressive decrease in blood pressure also in the jugular vein which, at times, reaches the magnitude on the order of minus 50 mm Hg. (Henry et al., 1951). Consequently, a certain blood pressure gradient is maintained in the arteries and veins which ensure blood flow in the vessels of the brain under the effect of the head-pelvis overload. However, one can hardly expect that a sufficient amount of oxugen could be supplied to the brain cells. There are indications in the literature which point to the development of the oxygen starvation of the brain under the effect of overloads in the cephalo-pelvic direction (Barr et al., 1958; Marshall et al., 1961, and others).

In the opinion of many researchers, oxygen starvation of the brain is the determining factor in withstanding the overloads in this direction (Shubert, 1937; Rosenblum, 1955; Malkin, Usachev, 1955). In order to study this problem further, we decided to use a method, which permits us to evaluate the oxygen supply to the brain during overloads using the magnitude of oxygen tension in the brain tissues.

PROCEDURE

The tests were conducted on dogs. First, platinum electrodes were implanted in the animal's brain into the cortex (motor region) and into the subcortex (thalmus) for determining the oxygen tension (pO_2) and recording of the EEG. The pO_2 was determined by the polarographic method. The oxygen tension was recorded in relative values (in%). Before the test, the O_2 tension was assumed to be 100% in the animal's brain while breathing air. The pneumogram, EKG, and EEG were recorded simultaneously with pO_2. Overloads from 2 to 12 g were produced by spinning the animals in a centrifuge. The animal was immobilized in the centrifuge in such a way that the centrifugal force, during the rotation, was directed from the head to the pelvis. Each load was exerted for one minute. A total of 59 tests were performed on 6 dogs.

RESULTS OF THE STUDY

In the first series of tests we studied the effect of overloads from 2 to 4 g. Throughout the entire period of rotation the animals were in the state of extreme agitation - barked and chewed at their restraints. Under these conditions, in all cases, we observed a slight increase in pO_2 (5–10% of the initial level) at the beginning of rotation of the animal. As the speed of rotation increased and a constant value of the overload was established (plateau), this increase was gradually replaced by a drop in pO_2 to the initial level and lower (Fig. 1). At an overload of 4 g, both the increase and decrease in pO_2 were more pronounced. The average pO_2 values were 97.6 and 92.9% at the 2 and 4 g, respectively. The change in oxygen tension occurred in an undulating manner. Particularly sharp fluctuations in pO_2 were observed during and at the end of the effect exserted by an overload of 4 g (Fig. 1).

Apparently, the undulating nature of the polarogram reflects the development of the compensatory reactions, especially the pressor mechanisms of the hemodynamics regulation which were directed towards maintaining the blood circulation in the brain under the effect of the inertial forces which caused an intensified shifting of blood from the head.

The compensatory reactions are also evidenced by an increase in the frequency of cardiac contractions (by 29 beats per 1 min at 2 g and by 41 beats at 4g) observed under these conditions. The respiration became less frequent but deeper. These compensatory reactions cause an increase in the blood flow, which leads to an incerase in pO_2 in the brain tissues in the initial period of action of small overloads.

In order to reveal the mechanism involved in the increase of pO_2 at the beginning of rotation, we conducted tests with the anesthetized animals, which were given 30 mg of barbamil and 2.0 cm^3 of 1% solution of morphia intramuscularly per 1 kg of body weight. In these tests, both the rise in pO_2 at the beginning of rotation and the fluctuations in pO_2 were less pronounced; at the loads of 4 g, we even observed a drop in pO_2 to below the initial level (Fig. 2). This, apparently, attested to the disruption of the compensatory reactions in the anesthetized animals due to the depression of the intercept reflexes and suppression of the cortical regulation of the hemodynamics and respiration.

The data obtained from the tests in which anesthetized animals were rotated make it possible to express a supposition regarding the reflex mechanism of the initial rise in pO_2 and the undulating nature of the polarogram under the effect of the overloads 2–4 g.

Based upon the results of all tests of this series, it is possible to conclude that the compensatory reactions of an organism can maintain oxygen tension in the brain tissues at a relatively high level during the overloads from 2 to 4 g, which act in the cephalo-pelvic position for 1 min.

In the next series of tests the magnitude of the overloads reached 6–8 g. At the beginning of rotation we observed, just as in the first series of tests, the excited movement of the animals which, however, was quickly replaced by depression. After the rotation stopped, the animals were kept stationary for 5–10 min. In 12 tests the rate of cardiac contractions increased and decreased in 4 tests. Usually, the respiration became less frequent; in one test at 8 g, we even observed a cessation of breathing.

The varied directionality of the reactions of the pulse and

respiration in the different tests indicates the difficulty of using these indices for the evaluation of the general state of an animal under the effect of accelerations. Towards the plateau of the overload of 8 g, slow, high-amplitude oscillations appeared on the EEG which lasted for 5–8 min after the rotation. The dogs withstood the overloads at 6 g better than at 8g.

The oxygen tension in the brain tissues at the beginning of rotation, just as in the first series, increased. However, this increase in pO_2 was brief and was quickly replaced by a sharp decrease, reaching a maximum towards the end of the overload's plateau. In most tests, the degree of decrease in pO_2 at 8 g was greater than that at 6 g. The average pO_2 values were 81.7% at 6 g and 72.4% at 8 g.

In isolated cases the oxygen tension continued to decrease after the centrifuge was stopped. Thus, in the dog named Tikhonya who has successfully withstood the overload of 6 g, we observed a sharp (up to 50%) decrease in pO_2 in the subcortex at 8 g, which lasted even after the rotation stopped. At the same time the dog stopped breathing, which was restored by an artificial respiration (Fig. 3). All this pointed to the fact that, at an overload of 8 g, the compensatory reactions of the organism can be suppressed not only during the rotation but also, for a certain period of time, after. A particularly grave state of the dogs was observed at the overloads from 10 to 12 g. In these tests, all dogs were depressed for 1 to 2 hours after the rotation; they reacted weakly to the external stimuli; during the rotation the respiration became less frequent and, sometimes, there was a complete cessation of the respiratory movements and cardiac contractions. The animals could be saved only by resorting to an artificial respiration. One dog perished despite the artificial respiration and the administration of the adrenaline and lobeline.

At the time of rotation the EEG showed slow high-amplitude oscillations, which could have been the predominace of the inhibitory process in the CNS.

At the overloads from 10 to 12 g the oxygen tension in the brain tissues reached the lowest values for these conditions of the

experiment, 76 to 67% on the average of the initial level.

The results of these experiments indicate the fact that when the overload acts in the head-pelvis direction, there is a natural decrease in oxygen tension in the brain tissues which, apparently, is caused by the disruption in the blood supply to the brain. In this case, impairment of external respiration observed in the experiments also plays a definite role.

The obtained results serve as a direct confirmation of the development of the oxygen starvation of the brain in animals under these conditions. However, at the overloads from 10 to 12 g, which cause drastic disorders in respiration and blood circulation, the level of pO_2 does not reach low values (on the average, up to 76.1–67.4%, respectively). At the same time, when an animal is lifted to an altitude of 12,000 m, where the conditions are right for the development of an acute oxygen starvation, severe hypoxic disorders were observed in dogs when the level of pO_2 in the brain tissues dropped by more than 50% of the initial level (Kovalenko, 1961). Consequently, the disorders which develop in an organism during acceleration cannot be attributed only to oxygen starvation of the brain.

We have all the reasons to agree with the assertion of a number of authors that, to a certain extent, these disorders are caused also by other factors, such as deformation of the organs and tissues, an intensive pathological impulsation from these organs and tissues, disruption of the regulating function of the cerebral cortex (Rosenblum, 1955; Savin, 1957; Sergeyev, 1957), and impaired gas exchange in the lungs (Ranke, 1937; Gauer, 1938).

In the combination with these factors, even a relatively small degree of hypoxy of the brain can play a significant role in the whole series of disorders caused by overloads.

CONCLUSIONS

1. A slight increase in oxygen tension of the brain tissues is observed in the initial period of overloads at 2–4 g in the cephalo-pelvic direction in 1 min of rotation which is then replaced by an insignificant decrease towards the end of rotation, to 97.6–93% on

the average. Under similar conditions, the decrease in the pO_2 level occurs without its initial rise in the anesthetized dogs.

2. At the overloads of 6–8 g the initial rise in pO_2 is quickly replaced by a decrease in oxygen tension, reaching 81–79% on the average.

3. Within the limits of these values, the more severe disruptions of the physiological functions were observed at the overloads from 10 to 12 g. Under these conditions we observed also the lower pO_2 values in the brain tissues, reaching 76–67% of the initial level on the average.

4. These results are a direct confirmation of the development of oxygen starvation of the brain under the effect of overloads on an organism.

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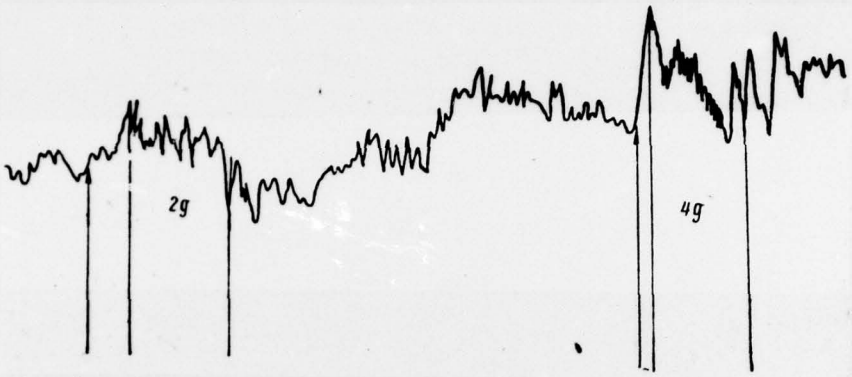

Fig. 1. Oxygen tension in the cerebral cortex of a dog under the effect of an overload from 2 to 4 g in the cephalo-pelvic direction.

Arrows - start of rotation; vertical lines - overload time.

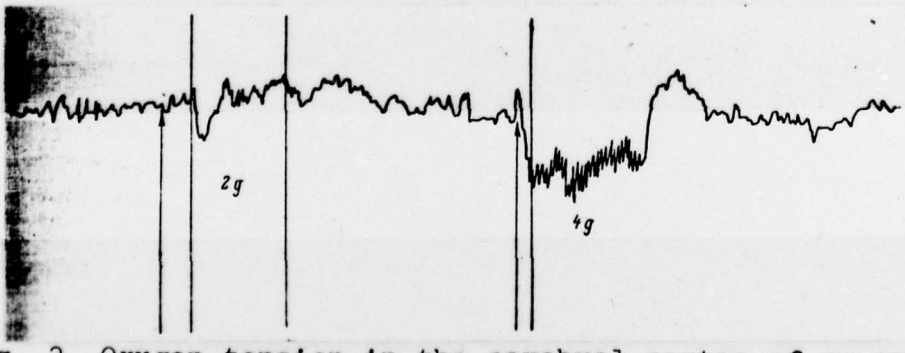

Fig. 2. Oxygen tension in the cerebral cortex of an anesthetized dog at an overlod from 2 to 4 g.

Designations are the same as in Fig. 1.

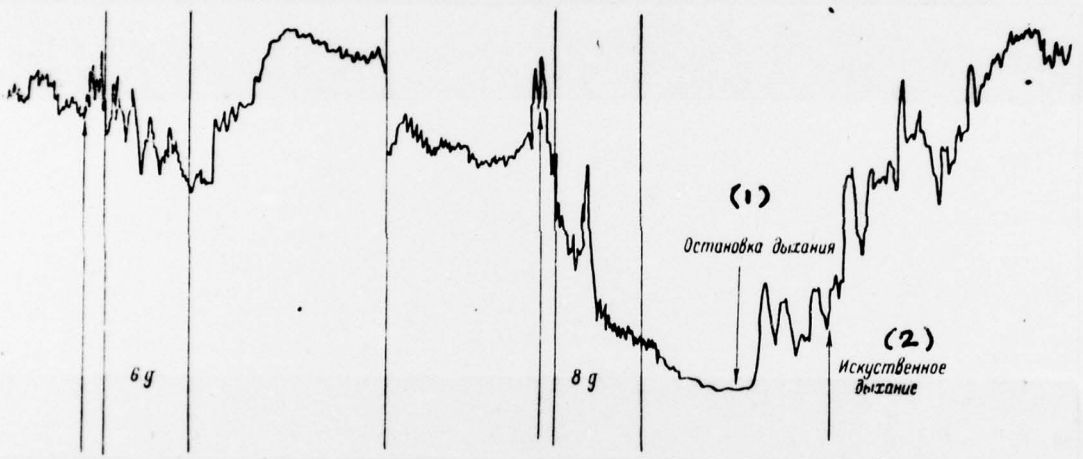

Fig. 3. Oxygen tension in the subcortex of a dog at the overloads of 6 and 8 g.

Designations are the same as in Fig. 1.

Key: (1) Cessation of breathing (2) Artificial respiration

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